



## **The Effects of Submaximal and Maximal Exercise on Heart Rate Variability**

CORY P. POEHLING\* and TAMRA L. LLEWELLYN†

Department of Health and Human Performance, Nebraska Wesleyan University, Lincoln, NE, USA

\*Denotes undergraduate student author, †Denotes professional author

---

### **ABSTRACT**

*International Journal of Exercise Science 12(2): 9-14, 2019.* The purpose of this study was to examine heart rate variability (HRV) at rest, and during submaximal (100 bpm) and maximal exercise in collegiate distance runners. We predicted there would be less HRV during exercise. Eight collegiate runners (19-22 yrs) were recruited for participation. The participants were equipped with a standard Lead II EKG to record HRV at rest. The participants then performed an incremental  $\text{VO}_{2\text{max}}$  test while running on a treadmill. EKG was recorded throughout the exercise test and HRV was later calculated during the submaximal and maximal exercise. To assess HRV the standard deviation of R-R intervals (SDNN) was calculated at rest and during submaximal and maximal exercise. A one-way ANOVA was used to determine HRV differences between these three states. The average R-R interval was  $0.961 \pm 0.155$  s (64 bpm),  $0.413 \pm 0.018$  s (146 bpm), and  $0.321 \pm 0.008$  s (187 bpm) for rest, submaximal, and maximal exercise, respectively. There were significant differences in SDNN from rest to submaximal ( $0.108 \pm 0.055$  to  $0.008 \pm 0.002$  s,  $p < 0.05$ ), and from rest to maximal exercise ( $0.108 \pm 0.055$  to  $0.006 \pm 0.002$  s,  $p < 0.05$ ). When comparing HRV between the resting and exercise states it seems that the parasympathetic nervous system (PNS) influence at rest contributes to greater HRV, whereas the sympathetic nervous system (SNS) influence during both submaximal and maximal exercise corresponds to a reduced HRV. These effects may be related to the enhanced automaticity effects of norepinephrine acting on its  $\text{B}_1$  receptor sites in the heart.

**KEY WORDS:** Autonomic nervous system; distance running; parasympathetic nervous system; sympathetic nervous system

### **INTRODUCTION**

It is often assumed heart rate consists of evenly separated beat-to-beat contractions. However, a unique feature the heart carries is its ability to beat at different paces, also known as heart rate variability (HRV) (3). High levels of HRV are considered a sign of autonomic flexibility and a good indication one will overcome when reacting to environmental changes and are associated with high levels of parasympathetic tone (2,4). On the other hand, low levels of HRV have been shown to increase vulnerability to chronic life stress, anxiety and mood states, and sudden cardiac death and are associated with low levels of parasympathetic tone (1,13). In this case, HRV is being used to predict future abnormalities in health by analyzing the autonomic nervous system (ANS) when working within the domains of frequency and time (11).

The parasympathetic nervous system (PNS) and sympathetic nervous system (SNS) work together to control and regulate different functions of the body including HR, therefore, producing HRV. The heart beats at an intrinsic rate of 100 beats per minute (bpm) because of the sinoatrial (SA) node (8). However, during rest, the PNS is active and slows HR down to an average rate of 60-80 bpm (6). Parasympathetic vagal nerves innervate the SA node on the heart releasing acetylcholine, causing bradycardia. Conversely, the nerves from the SNS acting on the SA node produce norepinephrine, causing tachycardia (8).

Much is known about HRV during rest; however, the effect of submaximal and maximal exercise intensities has yet to be examined in greater detail. Heart rate variability has been examined during cycle tests (7,9) and even in static and dynamic exercises (12) but none, to the author's knowledge, while running. The effect of submaximal and maximal exercise on HRV should not be assumed since its behavior during REM sleep is even unpredictable. Interestingly, during REM sleep, an unsuspected behavior occurs and an "activation" of the cardiovascular system results in lower HRV than even at wakeful stages (10). In other words, HRV mimics the SNS during sleep, when the PNS is active. The purpose of this experiment was to understand how HRV changes from resting to submaximal and maximal exercise in distance runners and how the ANS influences this variability. Due to the influence of the SNS during exercise, it is our expectation that as exercise intensity increases HRV will decrease compared to HRV at rest.

## METHODS

### *Participants*

The participants used for this research were from the Nebraska Wesleyan University Division III Track and Cross Country teams ranging from 18-22 years of age ( $n = 8$ ; Table 1). Exclusions from this research included any injuries limiting fluid range of motion while running, smoking within the prior year or the inability to complete the experimental design. Both men and women were included in this study (Men = 5; Women = 3). An informed consent was given and signed by the participants before data collection began. This study was approved by the Health and Human Performance Research Review Board at Nebraska Wesleyan University.

**Table 1.** Participant characteristics ( $n = 8$ ).

Descriptive	Men	Women
Age (yrs)	20.40 $\pm$ 1.14	20.33 $\pm$ 0.58
Height (cm)	178.4 $\pm$ 2.70	161.0 $\pm$ 10.15
Weight (kg)	69.78 $\pm$ 9.22	51.67 $\pm$ 1.44
VO <sub>2max</sub> (mL/kg/min)	61.02 $\pm$ 6.47	55.87 $\pm$ 3.84

\*Data are presented as mean  $\pm$  SD

### *Protocol*

This study examined the differences in HRV between submaximal, maximal exercise with HRV and resting HRV. The participants were connected to an electrocardiogram (EKG) machine to collect R-R intervals and completed a VO<sub>2max</sub> test on a treadmill. The R-R intervals were recorded

and interpreted for five minutes at rest, during the second minute of running (submaximal), and during the final minute of exercise before their maximal intensity was reached (maximal).

HRV was measured using a Biopac (MP35, Biopac Systems Inc., Goleta, CA). Three electrodes were placed over the thoracic cavity by using Einthoven's Triangle (Lead II). An electrocardiograph displayed the PQRST wave and represented beat-to-beat interactions of the heart. The time interval between the R-wave of the initial beat to the R-wave of the succeeding beat was recorded for five minutes to determine each R-R interval. The same procedure was carried out when the athletes were running at submaximal and maximal intensities during the  $\text{VO}_{2\text{max}}$  test (5). The participants began jogging at 8.0 mph (7:30 min/mi) and progressively increased their speed by a 1.0 mph after each two-minute interval. This continued until they reached their maximal intensity. If the athlete's terminal point was not reached in the first ten minutes, or if the treadmill no longer could produce the necessary speed for exercise, a grade inclination of 1% was added each minute after.

R-R intervals were calculated during the second minute of running and the final minute before reaching exhaustion during the  $\text{VO}_{2\text{max}}$  test. Finally, the standard deviation of R-R intervals (SDNN) was calculated for each of the three states to examine any differences in HRV between resting, submaximal, and maximal exercise states. Additionally, the difference between the largest and smallest R-R interval (MAX-MIN R-R) recorded was calculated for each of the three states.

$\text{VO}_{2\text{max}}$  was also obtained during this study using a metabolic cart (TrueOne 2400, Parvomedics, Sandy, UT). The participant was equipped with a mouthpiece, headgear, and nose clip and connected to a metabolic cart. The metabolic cart confirmed that participants were at or near max based on repeating  $\text{VO}_2$  values. Age-predicted maximal HR was also used to conclude if a participant had reached their maximal state.

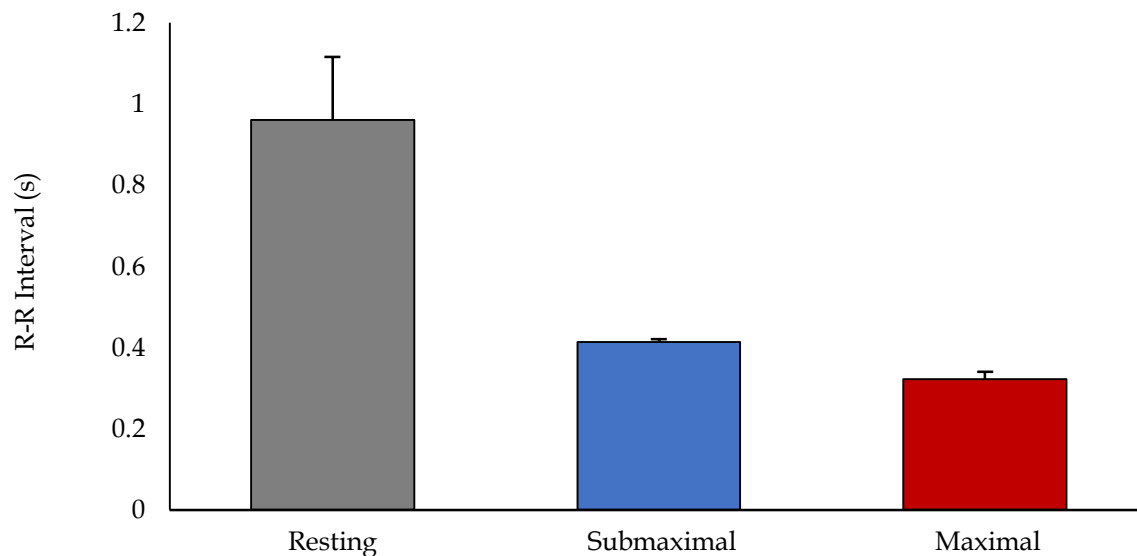
### *Statistical Analysis*

Data are presented as a mean  $\pm$  standard deviation. All HRV data were evaluated for differences between resting, submaximal, and maximal states by a one-way ANOVA. A Tukey's post-hoc test was used if significant differences occurred. Statistical significance was set at  $p \leq 0.05$ .

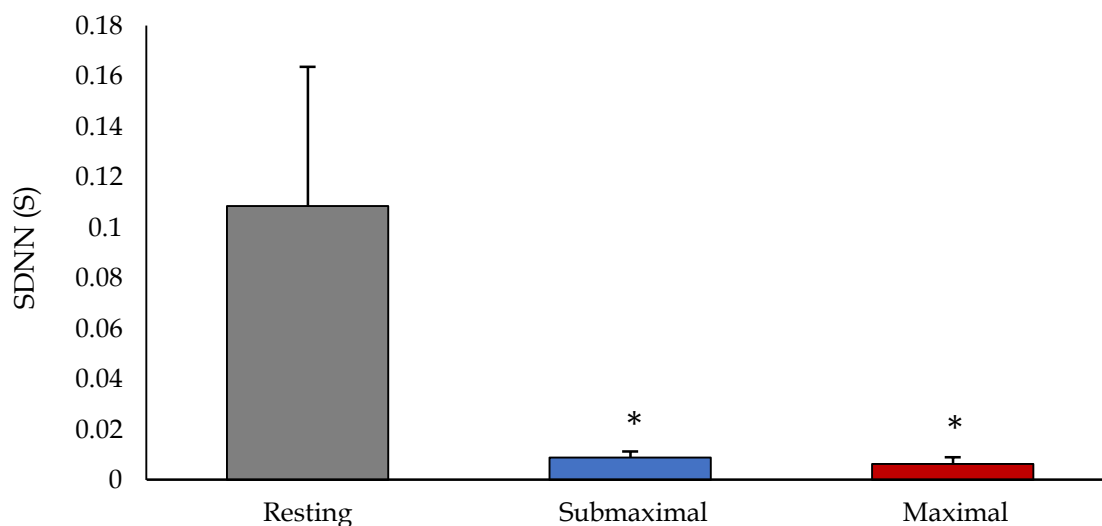
## **RESULTS**

The average R-R intervals at rest, submaximal, and maximal exercise were  $0.961 \pm 0.155$  s (64 bpm),  $0.413 \pm 0.018$  s (146 bpm), and  $0.321 \pm 0.008$  s (187 bpm), respectively (Figure 1).

As HR increases there is a noticeable decrease between R-R intervals. There were significant differences in SDNN from rest to submaximal ( $0.108 \pm 0.055$  s to  $0.009 \pm 0.003$  s,  $p = 0.047$ ), and from rest to maximal exercise ( $0.108 \pm 0.055$  s to  $0.006 \pm 0.002$  s,  $p = 0.047$ ; Figure 2). However, no significant differences were seen between submaximal and maximal exercise ( $0.009 \pm 0.003$  s to  $0.006 \pm 0.002$  s,  $p = 0.222$ ; Figure 2).

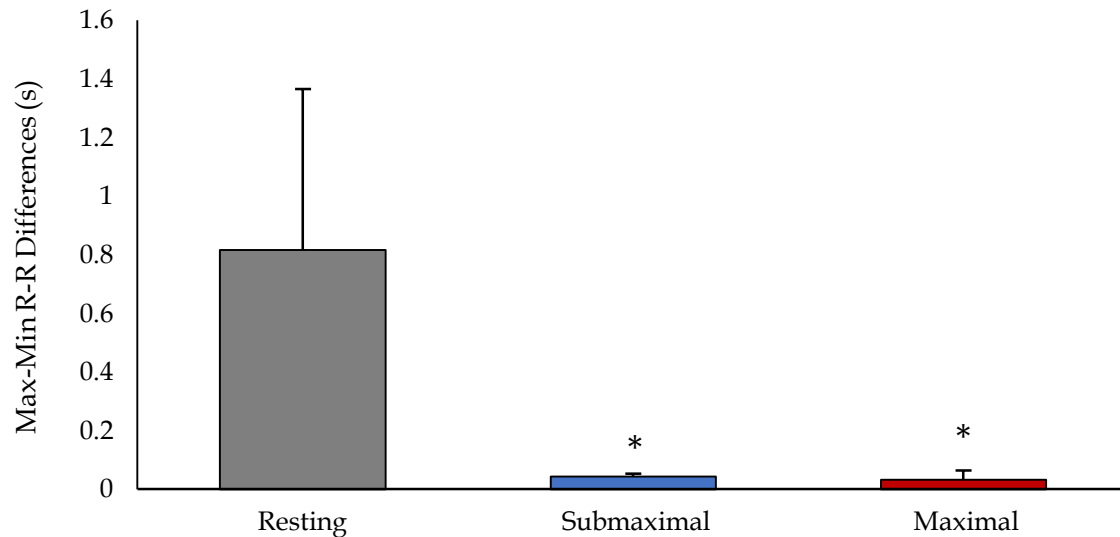


**Figure 1.** The effects of submaximal and maximal exercise on the R-R Interval. \* $p < 0.05$  vs. Resting.



**Figure 2.** The effects of submaximal and maximal exercise on SDNN. \* $p < 0.05$  vs. Resting.

Likewise, there were significant differences in Max-Min R-R differences from rest to submaximal exercise ( $0.817 \pm 0.549$  s to  $0.044 \pm 0.030$  s,  $p = 0.047$ ), and from rest to maximal exercise ( $0.817 \pm 0.549$  s to  $0.033 \pm 0.010$  s,  $p = 0.047$ ; Figure 3). However, like SDNN, there were no noticeable changes observed between submaximal and maximal exercise ( $0.044 \pm 0.030$  s to  $0.033 \pm 0.010$  s,  $p = 0.222$ ; Figure 3).



**Figure 3.** The effects of submaximal and maximal exercise on Max-Min Differences (s). \* $p < 0.05$  vs. Resting.

## DISCUSSION

The aim of this study was to examine the differences in HRV during exercise at submaximal and maximal intensities. In support of our hypothesis there wasn't an intensity-dependent effect on HRV, rather when running began, the PNS withdrawal and SNS activation produced a dramatic decrease in HRV at both submaximal and maximal exercise intensities compared to resting.

There were limitations to this study. Due to time constraints during the indoor track season, only eight participants could complete the study. A larger participant group would have emphasized the findings of this study. Another limitation of this study was that the wires, tubes, and straps being used during the  $\text{VO}_{2\text{max}}$  test were constraining to natural running. Ideally, wireless equipment would allow participants to run without any interference.

Finally, if true HRV were to be compared between the SNS and PNS, beta-blockers should have been used during the submaximal and maximal parts of the experiment to isolate the PNS, however, maximal exercise would have occurred much earlier. On the other hand, muscarinic receptor blockers could have been used during the resting condition to isolate the SNS. However, this was beyond the scope of this study.

As stated before, having an elevated HRV predicts one's ability to meet environmental stresses. On the other hand, having a lower HRV puts an individual at risk to abnormalities like cardiovascular disease (1). Since HRV is lower when under sympathetic innervation one area to question is whether this puts an individual at risk for irregular heart function during exercise. However, this interpretation is non-conclusive because no abnormal EKG readings were observed during testing and this study did not consider these kinds of EKG readings with great detail.

Additionally, if this area in HRV is studied in further detail in relation to exercise, the cardiac tissue would need to be examined closely to see how the  $M_2$  and  $\beta_1$  receptors contribute to receiving acetylcholine and norepinephrine, their respective neurotransmitters, from the sympathetic and parasympathetic nervous systems at different contraction rates. This may help explain in greater detail the differences seen when comparing HRV at different exercise intensities.

The results from this study suggest that at rest, there is a greater variability in HR in comparison to submaximal and maximal exercise, where HRV is practically non-existent. Unexpectedly, there were no differences in HRV between the two exercise intensities. The differences in HRV from rest to exercise likely occur at the cell-signaling level where during exercise, the activation of the  $\beta_1$  receptor on cardiomyocytes increases HR and automaticity of the heart. Further research at the receptor level regarding their signaling during exercise is needed to continue evaluation of these differences in HRV.

## REFERENCES

1. Carnevali L, Sgoifo A. Vagal modulation of resting heart rate in rats: the role of stress, psychosocial factors, and physical exercise. *Front Physiol* 5: 118, 2014.
2. Casadei B, Moon J, Johnston J, Caiazza A, Sleight P. Is respiratory sinus arrhythmia a good index of cardiac vagal tone in exercise? *J Appl Physiol* 81: 556-64, 1996.
3. Levy W, Cerqueira M, Harp G, Johannessen K, Abrass I, Schwartz R, et al. Effect of endurance exercise training on heart rate variability at rest in healthy young and older men. *Am J Cardiol* 82, 1998.
4. Liew R, Chiam P. Risk stratification for sudden cardiac death after acute myocardial infarction. *Ann Acad Med Singapore* 39, 2010.
5. McNarry M, Lewis M. Heart rate variability reproducibility during exercise. *Physiol Meas* 33, 2012.
6. Mohrman D, Heller L. Cardiovascular physiology. New York: McGraw Hill Lange; 2010.
7. Pichon AP, De Bisschop C, Roulaud M, Denjean A, Papelier Y. Spectral analysis of heart rate variability during exercise in trained subjects. *Med Sci Sports Exerc* 36: 1702-8, 2004.
8. Rowell L. Human circulation regulation during physical stress. New York: Oxford University Press; 1986.
9. Sarmiento S, García-Manso JM, Martín-González JM, Vaamonde D, Calderón J, da Silva-Grigoletto ME. Heart rate variability during high-intensity exercise. *J Syst Sci Complex* 26: 104-16, 2013.
10. Tobaldini E, Nobili L, Strada S, Casali KR, Braghiroli A, Montano N. Heart rate variability in normal and pathological sleep. *Front Physiol* 4: 294, 2013.
11. Tonello L, Rodrigues FB, Souza JW, Campbell CS, Leicht AS, Boullosa D. The role of physical activity and heart rate variability for the control of work related stress. *Front Physiol* 5: 67, 2014.
12. Weippert M, Behrens K, Rieger A, Stoll R, Kreuzfeld S. Heart rate variability and blood pressure during dynamic and static exercise at similar heart rate levels. *PLoS One* 8: 1-8, 2013.
13. Wood SK. Cardiac autonomic imbalance by social stress in rodents: understanding putative biomarkers. *Front Psychol* 5: 950, 2014.